DIP #596

2025 USMLE Step 1 Free 120 Discussion Part 11b
(Q106-110, super helpful for Step 2 and 3!)

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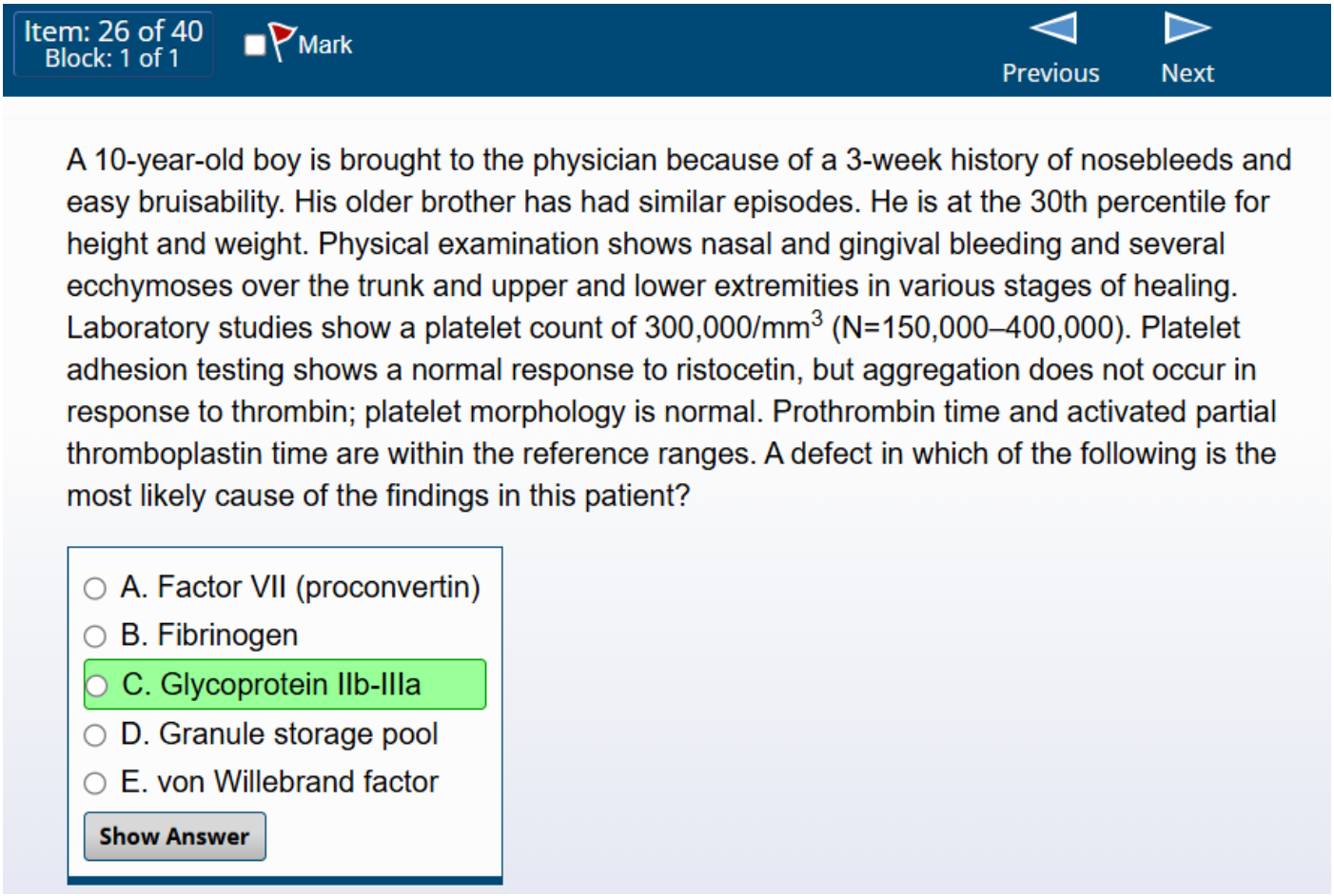
- **Dr Mohammed Zaki**
https://chat.whatsapp.com/L6dShASc3f97cwHZgCD6Xo

Welcome.

This is Episode 596 of the Divine Intervention Podcast. In this podcast, we're going to be continuing the Step 1 Free 120 series. This is going to be Series 11B.

Question 106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A 10-year-old boy is brought to the physician because of a 3-week history of nosebleeds and easy bruisability. His older brother has had similar episodes. He is at the 30th percentile for height and weight. Physical examination shows nasal and gingival bleeding and several ecchymoses over the trunk and upper and lower extremities in various stages of healing. Laboratory studies show a platelet count of 300,000/mm3 (N=150,000–400,000). Platelet adhesion testing shows a normal response to ristocetin, but aggregation does not occur in response to thrombin; platelet morphology is normal. Prothrombin time and activated partial thromboplastin time are within the reference ranges. A defect in which of the following is the most likely cause of the findings in this patient?
A. Factor VII (proconvertin)
B. Fibrinogen
C. Glycoprotein IIb-IIIa
D. Granule storage pool
E. von Willebrand factor
Show Answer

This is a very simple question if you understand primary hemostasis. Remember, primary hemostasis has a bunch of steps. So the very first step is the adhesion step, where GP1b binds von Willebrand factor. And then after that, you have the activation step, where after GP1b binds to von Willebrand factor, the platelet secretes ADP, and then that ADP binds to the ADP receptor, which sometimes on exams is called the P2Y12 receptor. And that triggers the third step.

So when ADP binds to its ADP receptor, it's almost like an autocrine secretion in a sense. Then you're going to express GP IIb/IIIa, and then GP IIb/IIIa from one platelet will bind to GP IIb/IIIa from another platelet, and they're going to be joined together by fibrinogen. That's going to be the aggregation step. So step number one is adhesion, step number two is activation, and step number three is aggregation.

So one thing that's pretty high-yield to know for your exams is the ristocetin test. The ristocetin test—sometimes it's called the ristocetin cofactor assay—is an excellent way to test for the integrity of the adhesion step of primary hemostasis. So the thing is, if you notice, they say platelet adhesion testing shows a normal response to ristocetin. So that

pretty much rules out von Willebrand disease, because remember von Willebrand disease is absolutely an adhesion problem. It's a deficiency of von Willebrand factor. It's autosomal dominant.

And then the other one, Bernard-Soulier disease—that's a GP1b deficiency. That one is autosomal recessive. If you notice here, this person has normal platelet adhesion testing. So the response to ristocetin is, we're told, normal. So this cannot be von Willebrand disease or Bernard-Soulier disease.

Remember, von Willebrand disease will also cause your PTT to rise, because von Willebrand factor protects factor VIII. But in Bernard-Soulier, your PT and PTT will be fine because GP1b has nothing to do with clotting factors.

So we're told that aggregation does not occur in response to thrombin. So this has to be a platelet aggregation defect. And remember, aggregation is GP IIb/IIIa. So this person probably has Glanzmann thrombasthenia.

Remember, Glanzmann disease is an autosomal recessive disease where you have a deficiency of GP IIb/IIIa. It's a qualitative—not a quantitative—platelet defect. If you notice, the platelet count here is normal. It's just the platelets do not work very well because they lack GP IIb/IIIa. So it's a GP IIb/IIIa deficiency.

So the answer has to be C.

In option A, Factor VII proconvertin—that's wrong. A Factor VII defect you're going to see in things like liver disease. Your liver is a very important producer of Factor VII. When people have liver failure, we're really worried about the Factor VII levels. So that can cause them to have a lot of bleeding. Remember, Factor VII is part of secondary hemostasis, so you're going to see abnormalities at least with the PT. But here we're told that the PT and PTT are completely normal, so that's wrong.

Option B says fibrinogen. Again, fibrinogen is what joins one GP IIb/IIIa to another GP IIb/IIIa. But again, typically on the USMLEs, they love to test Glanzmann thrombasthenia, so we're going to go with that.

And then granule storage pool—that's wrong.

And then von Willebrand factor—that's wrong, and I explained why that's the case.

And please, again, don't forget that when a person has Glanzmann thrombasthenia—because it's a primary platelet disorder—their bleeding time is going to be elevated. But again, GP IIb/IIIa has nothing to do with clotting factors, so your PT and PTT will be fine.

Bernard-Soulier disease is a GP1b deficiency. It's a primary platelet disorder, so your bleeding time will be up, but your PT and PTT should be fine because GP1b does nothing with clotting factors.

And then for von Willebrand disease—it's a primary platelet disorder, so your bleeding time is going to be elevated. But your PTT will be elevated because, again, von Willebrand factor increases the half-life of Factor VIII in circulation. So if von Willebrand factor is not around—because you have a deficiency of it—you will not be able to increase the half-life of Factor VIII in circulation. So you're going to have a high PTT. Your PT should be unaffected.

So you may wonder, “Man, Divine, how do I differentiate von Willebrand disease, which is an adhesion defect, from Bernard-Soulier disease, which is also an adhesion defect?”

Well, the thing is, both of those disorders will have increased bleeding time. But von Willebrand disease will have an increased PTT, and Bernard-Soulier will have a normal PTT.

And remember that those are both adhesion problems, so you're going to have abnormalities with the ristocetin cofactor assay. So the ristocetin cofactor assay is abnormal both in Bernard-Soulier disease and in von Willebrand disease. So please don’t be surprised if they give you a question about a person who has gum bleeds and all these things, PTT is high, bleeding time is up, and then they say, “Which of the following additional lab studies will be abnormal in this patient?” Pick the answer choice that says ristocetin cofactor assay. That’s actually a very nice way they can test that material on the exams.

Question 107

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A previously healthy 45-year-old woman who works as a park ranger comes to the physician because of a 1-week history of shortness of breath, even at rest. She has lived in the mountains at 10,000 feet above sea level for 2 years; the physician's office is located at sea level. Her pulse is 85/min, respirations are 18/min, and blood pressure is 125/90 mm Hg. Physical examination while sitting upright shows jugular venous distention and 2+ pedal edema. During the past 2 years, which of the following has most likely decreased in this patient?

- A. Height of P waves in lead I of the patient's ECG
- B. Height of R waves in lead V_1 of the patient's ECG
- C. Hematocrit
- D. Pulmonary vascular resistance
- E. Right ventricular diastolic compliance
- F. Right ventricular wall thickness

Show Answer

So, this person has been living at very high altitudes, right? Let's work this out—even at very high altitudes, we know that the oxygen content in the atmosphere is lower. Not because your FiO_2 is low—your FiO_2 is still the same at sea level or at higher elevations.

The only thing is that your atmospheric pressure has decreased. So, because your atmospheric pressure has decreased, your oxygen—21% of air -you're taking in 21% of a lower atmospheric pressure. So overall, that's going to decrease the oxygen content.

It's like the difference between taking 21% of 760 mmHg versus 21% of 700 mmHg. Just something to keep in mind.

So, this person will probably have some degree of hypoxia. We're going to talk about some of the changes that can happen with some of these answers here.

So, option A says height of P waves in lead I of the patient's ECG. So that's not going to be the case. And honestly, options A and B are a very extensive discussion that I will very likely make in a future podcast. Again, I don't know when that future podcast will be, but I'm very likely to make it.

But this person, if you think about it—just to give you a really small, brief preamble—this person probably has, from living at high elevations, hypoxia from the decreased oxygen content in the air. That is going to decrease the person's PAO_2—that's the alveolar oxygen tension. So obviously, because that is low, the PaO_2—that's the arterial oxygen tension—will be low as well.

That hypoxia is going to cause pulmonary vasoconstriction. When your pulmonary vessels constrict, it's going to become harder for blood to be ejected from the right side of the heart. If that happens, your right ventricle will get big and beefy. You will develop pulmonary hypertension. So the right side of your heart is going to enlarge. You're going to have right atrial, right ventricular hypertrophy and stuff like that. Your right atrium is going to enlarge.

And typically, if your right atrium enlarges, you're going to see kind of tall P waves in leads II, III, aVF—those are your right-sided leads—and also in leads V1 and V2. And also the height of the R waves in lead V1 will go up. So honestly, A and B are wrong. I know I'm just giving a very brief explanation. Honestly, it's just a much more extended discussion. Now is not a good time.

I just want to go through this series—now is not a good time—because honestly, to really understand EKGs in that depth requires a very extensive discussion, and I just don't think this podcast is the appropriate forum for that. Again, that's a topic for a future podcast. So just believe me for now: A and B are wrong.

Option C says hematocrit. Hematocrit should actually be increasing in this person. Because if you think about it, that hypoxia causes pulmonary vasoconstriction. And also—so I guess let me answer option D—it causes pulmonary vasoconstriction.

So because you're getting this vasoconstriction of your pulmonary vessels, your pulmonary vascular resistance should go up, because those vessels are constricted. But think about it—when you have hypoxia, what happens to your EPO? Your EPO is going to rise because we want to increase the number of red cells. So when you increase that number of red cells, that's going to raise your hematocrit. So your hematocrit is not going to be decreasing.

And then option E says right ventricular diastolic compliance. Well, think about it—when you develop pulmonary hypertension, that's going to cause chronic pressure overload on the right ventricle. So the right ventricle is going to get very big and beefy. So because it's getting big and beefy, you're adding sarcomeres in parallel. It's going to become harder and harder for the right ventricle to expand because it's just gotten very big and beefy. It's not as compliant as before.

I almost think of it as being very similar to what you have in restrictive lung disease, like pulmonary fibrosis, where it's like now you've deposited so much stuff in the walls of your lungs that they don't expand. Here, the thing you've deposited a ton of is muscle. So it's like you're RV is so muscular, you cannot expand very well. It's kind of stiff, so it's not as compliant. So your right ventricular diastolic compliance would certainly go down. So option E is definitely the answer.

And then option F says right ventricular wall thickness. That has to be wrong, because again, I just explained why your right ventricle should get big and beefy. So your right ventricular wall thickness should actually increase. So option F is wrong.

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A 5-year-old girl is brought to the office by her mother because of a 6-hour history of bloody diarrhea. She is interactive and in no acute distress. Her blood pressure is 90/55 mm Hg. Abdominal examination shows normoactive bowel sounds. Stool cultures are obtained, and the patient's mother is advised to give the girl plenty of fluids. Five days later, the patient develops decreased urine output and is brought back to the office. Her blood pressure is now 135/88 mm Hg. Physical examination shows pallor. Laboratory studies show:

Hemoglobin	8.5 g/dL (N=11–15)
Hematocrit	26% (N=28%–45%)
Platelet count	45,000/mm^3 (N=150,000–400,000)
Serum creatinine	3.3 mg/dL (N=0.3–0.7)

Which of the following infectious agents is the most likely cause of these findings?

- A. *Campylobacter jejuni*
- B. *Escherichia coli*
- C. Rotavirus
- D. *Salmonella enterica* serovar *enteritidis*
- E. *Yersinia pestis*

Show Answer

So, we see a child that has had bloody diarrhea. And then we also notice that this person is now having anemia, thrombocytopenia, and acute kidney injury. This is a pretty classic presentation of Hemolytic Uremic Syndrome—HUS.

And remember, there are two classic causes of HUS on the exam. There's going to be E. coli O157:H7, and then there's going to be Shigella, although E. coli O157:H7 is a far more common cause of HUS than Shigella.

So the answer here has to be option B.

Remember, option A: Campylobacter jejuni causes bloody diarrhea, but it generally does not cause HUS.

Rotavirus is the most common cause of infectious gastroenteritis in kids, but the diarrhea is usually watery, not bloody. So that's wrong.

Now, Salmonella also causes bloody diarrhea, but again, Salmonella typically does not cause HUS on the exams.

And then option E says Yersinia. Yersinia pestis, I believe, is what causes the bubonic plague. Remember, there is Yersinia enterocolitica, which can cause pseudoappendicitis and can also cause bloody diarrhea. But that's Yersinia enterocolitica—not Yersinia pestis. I believe Yersinia pestis is what causes the bubonic plague. So that's wrong.

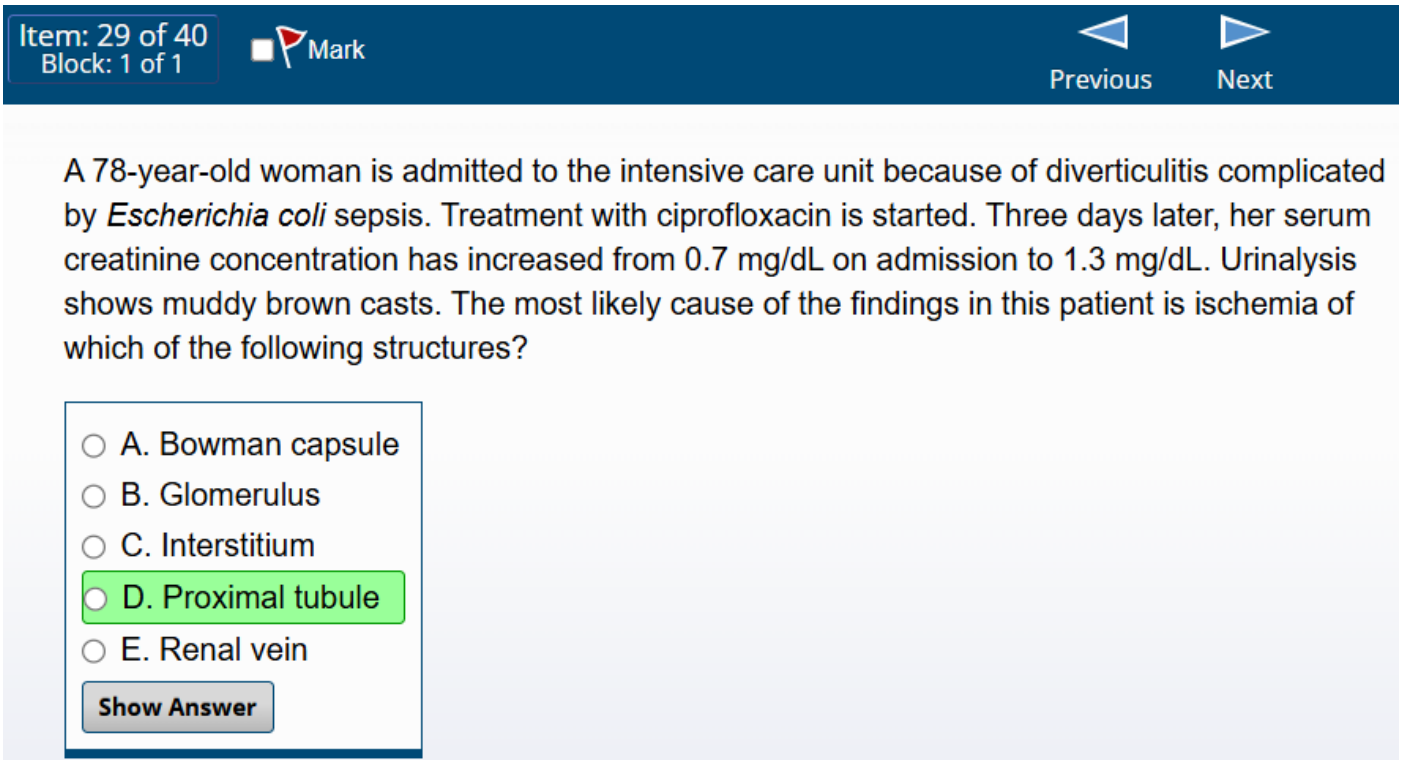
This is not the bubonic plague—I can tell you that.

So again, what are the components of HUS? You're going to see fever, anemia—and the anemia is usually going to be a microangiopathic hemolytic anemia. So you're going to see schistocytes on a blood smear. Sometimes they'll have indirect hyperbilirubinemia because of all that hemolysis. You'll have thrombocytopenia. They have renal failure.

And typically, the treatment is just supportive. You actually do not want to give antibiotics when a person has HUS. Because here's the thing—if you give antibiotics, that's going to cause a huge ruckus for the person because it's the toxin that's causing the disorder. So if you give antibiotics, you kill more bug. If you kill more bug, you release more toxin. If you release more toxin, you will cause more damage—for the kidneys and stuff like that. So don't give antibiotics. It's not every infection that should get antibiotics.

So the answer for this question is option B. E. coli O157:H7 is the most common infectious cause of HUS.

Question 109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A 78-year-old woman is admitted to the intensive care unit because of diverticulitis complicated by *Escherichia coli* sepsis. Treatment with ciprofloxacin is started. Three days later, her serum creatinine concentration has increased from 0.7 mg/dL on admission to 1.3 mg/dL. Urinalysis shows muddy brown casts. The most likely cause of the findings in this patient is ischemia of which of the following structures?
A. Bowman capsule
B. Glomerulus
C. Interstitium
D. Proximal tubule
E. Renal vein
Show Answer

This person probably has **acute tubular necrosis (ATN)**. We can see muddy brown casts, we can see this rapid rise in creatinine. So this is ATN.

Although, let me say something here. You want to be careful—sometimes on the USMLE exams, instead of seeing the term *acute tubular necrosis*, you may actually see the term *acute tubular injury*. So just be careful about that. They use the term *acute tubular injury*.

And the thing is, acute tubular necrosis can have many different causes. Sometimes they even call it "toxic ATN." Usually, that's going to be from a drug that's nephrotoxic, like **vancomycin** or an **aminoglycoside**, or things like **cisplatin**. Remember, platinum agents are pretty nephrotoxic. Or even **loop diuretics**—they can be nephrotoxic, especially **ethacrynic acid**. And then remember **myoglobin**, like in the setting of rhabdomyolysis, or **hemoglobin**, like in the setting of some kind of acute hemolytic transfusion reaction—those things can all cause ATN.

But then there is **ischemic ATN**, which usually arises from a **hypoperfusion state**. So, say for example, a person has an MI, and they're not perfusing the kidneys, or a person has just had some kind of issue that's making them not perfuse the kidneys—well, that can cause ischemic ATN.

But basically, the sum total of all of this is: your kidneys get messed up. So they won't be able to do their reabsorption functions very well. So the fractional excretion of sodium will be high—it will usually be **more than 3%** on your exams.

So the key thing is: **who gets affected the most**?

Well, the thing is, if your kidneys are being torched by a nephrotoxic agent or you have hypoperfusion of your kidneys, the parts that use up the most energy are going to be the most heavily affected.

So what parts of your kidneys use the most energy?

Well, number one is your **proximal tubule**. That's why **option D is the answer**.

But another part of your kidney that uses a ton of energy is the **thick ascending limb of the loop of Henle**. That's actually pretty high-yield to know for your exams. The thick ascending limb of the loop of Henle—that's in the medulla—those are the ones that use the most energy.

And you may wonder, "Divine, why does the proximal tubule use so much energy?" Well, that should make sense. Think about it—it reabsorbs like **most of the stuff that is filtered** into your urine. So just something to keep in mind.

So the answer is going to be **option D**.

Honestly, none of the other answers make any sense here.

Remember, the **glomerulus** is what is damaged in a person that has **nephritic syndrome**.

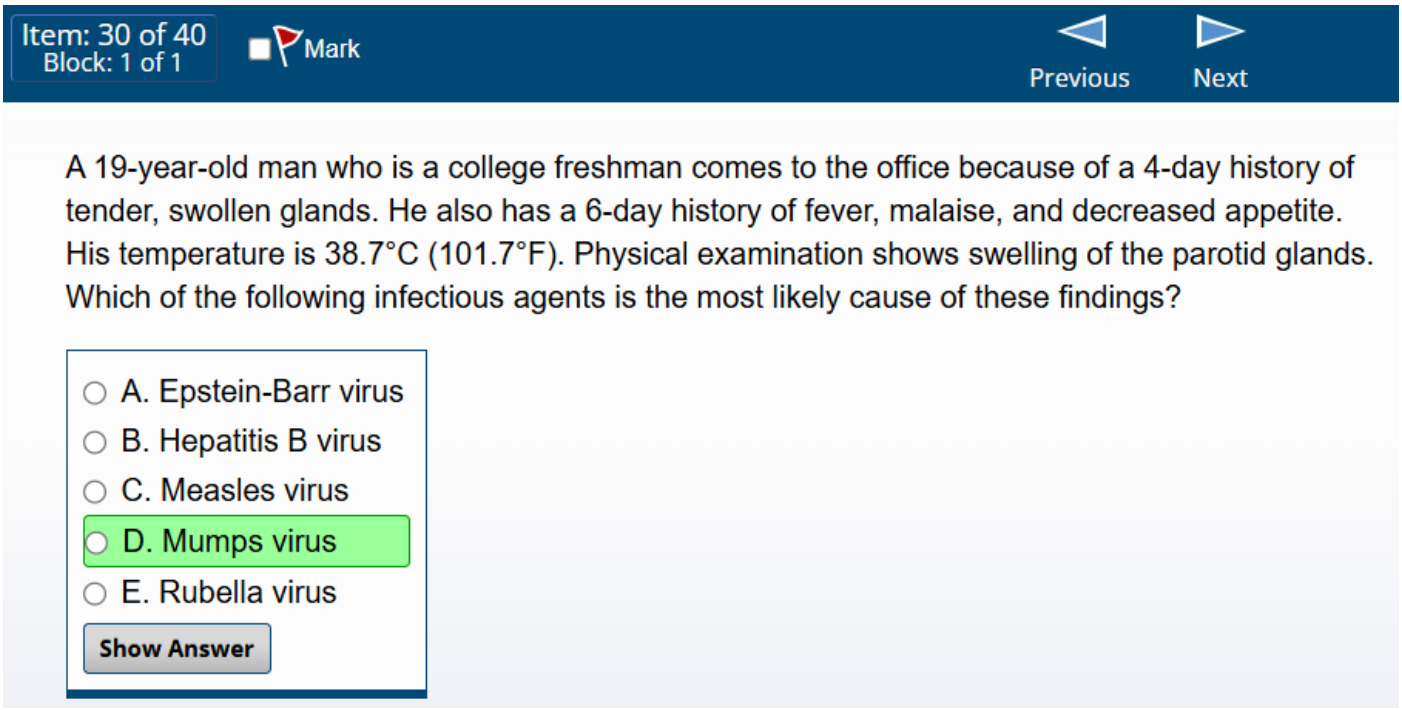
The **interstitium** is what is damaged in **acute interstitial nephritis**, which usually has that triad of **fever, rash**, and **increased eosinophils** in the blood, plus or minus the urine. And typically that's going to be **after antibiotic exposure**, like NSAID exposure.

Renal vein—remember, you can have renal vein thrombosis when you have **nephrotic syndrome**, especially **membranous nephropathy**. Because remember, in nephrotic syndrome, one of the things you lose in your urine is—oh come on Divine, think—you lose **antithrombin III**.

Nephrotic syndrome can cause an acquired **antithrombin III deficiency**, and antithrombin III is an **anticoagulant protein**. Because it inhibits **Factor II** and **Factor X**. So if you lose that—if you don't inhibit Factor II and Factor X—they're going to have a longer half-life. They're going to make you **hypercoagulable**, and you're going to get in trouble. So actually, there's a very nice question there.

So that's why **renal vein thrombosis** is a pretty common phenomenon in nephrotic syndrome—especially of **membranous nephropathy**. Membranous nephropathy is probably—not probably, it's almost certainly—the most common kind of nephrotic syndrome that's associated with renal vein thrombosis.

Question 110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A 19-year-old man who is a college freshman comes to the office because of a 4-day history of tender, swollen glands. He also has a 6-day history of fever, malaise, and decreased appetite. His temperature is 38.7°C (101.7°F). Physical examination shows swelling of the parotid glands. Which of the following infectious agents is the most likely cause of these findings?
A. Epstein-Barr virus
B. Hepatitis B virus
C. Measles virus
D. Mumps virus
E. Rubella virus
Show Answer

So this is pretty straightforward. We see a person that has **parotitis**. This bug also causes **orchitis**—causes testicular inflammation. And this bug can also cause **acute pancreatitis**, by the way.

This is **pretty classic mumps**.

So the answer is **D**.

Remember, **orchitis can cause infertility**—just be careful about that. And remember, **mumps** is an important cause of **acute pancreatitis** on the USMLEs.

This is not **EBV**—they're going to have a sore throat, they can have hepatosplenomegaly. That's not what's going on here.

Hepatitis B—in this case, they would have probably given us abnormal LFTs. They would be giving us these **HBV serologies** or something about needle-stick injury or sexual transmission or blood transfusion or something like that—we don't see that here.

Remember **measles**—measles is going to present with **cough, coryza** (that's runny nose), and **conjunctivitis**. We don't see that here.

And then **rubella**—rubella, you're going to have this **rash** that starts at the top of your head and comes downward. And many times you have **posterior auricular lymphadenopathy**. Again, we don't see that here. So rubella is wrong.

So the answer is **option D**. This is the **mumps virus**.